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Peronosclerospora Sacchari

Department of Agriculture,
BRISBANE,
QUEENSLAND.

BUREAU OF SUGAR EXPERIMENT STATIONS.

TECHNICAL COMMUNICATIONS.

YEAR 1941, No. 5.

Downy-Mildew Disease of Sugar Cane and Other Grasses.

by

C. W. LEECE.

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SUMMARY.

Downy-mildew disease of sugar cane caused by *Sclerospora sacchari* T. Miy., occurs in Queensland, Formosa, Fiji, the Philippine Islands, India and possibly in New Guinea and Siam. It was first reported in a technical publication in 1911, from Formosa, where it was found attacking sugar cane that had been imported from Queensland; it has never been observed to attack any indigenous grass in this country, and the country of origin is still unknown.

The symptoms of the disease on sugar cane are extremely diverse and depend largely on the nature of the infection (i.e., whether primary or secondary), and the stage in the life cycle of the causal fungus. The asexual stage and its concomitant features are the more common, and under favourable meteorological conditions (such as occur in Queensland between September and May), there develop characteristic leaf streaks accompanied by the nocturnal production of a fine white "mildew" consisting of conidia and conidiophores of the parasite. An oospore stage may develop in the winter; this is characterised by the abnormal elongation of stalks and fine shredding of the leaves.

Transmission is effected by conidial infection of immature nodal buds. The conidia are extremely delicate and remain viable for only a few hours, even under the most favourable conditions. Oospore infection has not yet been demonstrated. The planting of diseased setts invariably gives rise to diseased stools although all setts taken from diseased stools are not necessarily infected; young plants are more easily infected than older ones.

The relation of *S. sacchari* to various crop and grass plants has been investigated: maize and teosinte both proved to be highly susceptible; symptoms on these hosts differed markedly from those exhibited by sugar cane and sorghum. Diseased maize plants were often extremely malformed and yields were greatly reduced; mycelium of the causal fungus was traced into the seed, but seed from diseased plants produced healthy seedlings in the plantings made. Abundant conidia were produced under favourable conditions but oospore development was never observed.

The disease was also transmitted to sorghum, although this crop is fairly resistant to infection; in this case conidial production was always sparse and oospores were never observed. Johnson grass (*Sorghum halepense* (L.) Pers.) and Sudan grass (*Sorghum sudanense* Stapf) gave indications of being able to function as alternative hosts of *S. sacchari*, as judged by the appearance of leaf markings on plants exposed to infection. A doubtful case of conidial production was recorded on Johnson grass on one occasion only; oospores were never found. Further work on these two possible hosts, under more tropical conditions, is desirable.

The following grasses all failed to contract the disease:—*Panicum maximum* Jacq., var. *coloratum* (purple-topped Guinea grass), *P. maximum* Jacq. (Guinea grass), *Brachiaria purpurascens* (Raddi) Henrard (Para grass), *Echinochloa frumentacea* Link. (Japanese millet), *Pennisetum purpureum* Schum. (Elephant grass), *P. typhoides* Stapf and Hubbard (Pearl millet), *Chloris gayana* Kunth. (Rhodes grass), *Sorghum vulgare* Pers. (Egyptian wheat), and *Erianthus arundinaceum* (Retz.) Jesw.

Transmission of the disease from maize to sugar cane was effected under controlled conditions.

Downy-Mildew Disease of Sugar Cane and Other Grasses.

by

C. W. Leece.

1. Downy-Mildew Disease of Sugar Cane.

History and Distribution.

It is not known in what part of the world downy-mildew disease first attacked sugar cane, nor whence it came to Australia, but the fact that it has not yet been found on any indigenous grass of this country suggests that it was introduced here from some outside source. Towards the end of the last century a number of consignments of sugar cane were imported from New Guinea, and it is possible that one of these introductions is responsible for its presence. It is true that the causal fungus, *Sclerospora sacchari* T. Miy., has not yet been specifically reported from New Guinea, but Lennox [**18,** 1938] has observed a downy-mildew disease on *Saccharum spontaneum* growing along the headwaters of the Purari River. Moreover, New Guinea still possesses vast tracts of unexplored territory, and a comprehensive survey would probably yield much information of phytopathological interest.

The genus *Sclerospora* has world-wide distribution, but records of *S. sacchari* as a parasite of sugar cane are confined to a few countries in the Western Pacific region. Other species, closely related to *S. sacchari* from the standpoints of morphology and host range, are known to occur in Java, the Philippine Islands, Formosa and India; these likewise have a limited geographical range. *S. sacchari* differs markedly from any of the species described from Europe or North America, so that it is logical to assume from the present known facts that its origin may be traced to somewhere in the above-named regions.

In 1901, in the Herbert River District of North Queensland, there occurred an outbreak of a "new" disease, and from records of conversations with farmers in the district it would appear that this disease was downy mildew. Be that as it may, downy mildew was definitely known to be present in Queensland by 1910, and was at that time widely distributed throughout the Northern and Central districts. Canegrowers did not regard it as an infectious disease, but considered it to be a varietal peculiarity that had arisen as a result of deterioration of the affected varieties—brought about by continuous vegetative propagation, or other more obscure causes.

North [**30,** 1910-15] commenced observations on this disease in the Herbert River District in 1910, and established the fact that it was an infectious fungous disease and that it was spreading rapidly; it was then locally known as "rust" but later it became known as "leaf stripe." It is still widely distributed in Queensland cane-growing areas, but has never been observed in districts south of Bundaberg.

In addition to Australia, the disease has been recorded in Formosa, Fiji, the Philippines, India, and possibly New Guinea and Siam. It was first observed in Formosa in 1909, attacking cane in fields of the Formosan Government Sugar Experiment Station. The infected varieties had been recently introduced into Formosa from Queensland and Java, but although a species of *Sclerospora,* (*S. javanica*) is known to occur in Java, downy mildew of sugar cane has not been reported from that country; the Queensland importations must therefore be considered responsible for the introduction of the disease into Formosa. Miyake [**28,** 1911] carried out an investigation of the disease and named the causal fungus *Sclerospora sacchari*; he found that cuttings from infected plants always result in diseased stools; that the fungus spores may be carried from plant to plant by wind, rain, and irrigation water; and that maize and teosinte may function as alternative hosts of the parasite; at the same time he was unable to infect sorghum or millet.

Downy mildew was known to be present in Fiji in 1911, and a brief description of symptoms was made by Lyon [**20,** 1915] who recognised the disease as being identical with that described by Miyake.

A shipment of canes from Formosa to the Philippines was followed by the discovery of the disease in the latter country by Lee and Medalla [**17,** 1921] in 1920. Steps were taken to plough out and destroy the infected cane, although some of it had already been distributed. The disease is reported by Ocfemia to be still present in the Philippines [**31,** 1938] but it does not appear to have caused any serious losses. In this connection, however, it should be noted that Miyake (*see* Cook [**7,** 1932]) is of the opinion that the *Sclerospora* reported by Lee and Medalla was not *S. sacchari,* but *S. spontanea.* Two species of *Sclerospora* (*S. spontanea* and *S. philippinensis*), known to occur in the Philippines, resemble *S. sacchari* very closely in morphological features, but do not appear to be strongly pathogenic on sugar cane—or at least on those varieties of cane which are grown commercially in these islands.

Downy mildew of sugar cane was discovered on the variety Co.316 at Pusa, India, by Mitra [**27,** 1931] in 1930. Subramaniam [**43,** 1931], however, believes that the Indian species of *S. sacchari* differs from that of Formosa, although the conidial measurements which he made fell within the limits of those described by Miyake. As in the Philippines, prompt measures were taken to deal with the outbreak, and judging from the absence of further references, the disease may have been entirely eliminated. None of the available reports states to what source the outbreak at Pusa may be attributed, although this was the first record of the disease in India.

Stevenson and Rands [**41,** 1938] and Cook [**7,** 1932] list *S. sacchari* as being present in Siam but the authority for the record is not cited in either compilation.

Symptoms on Sugar Cane.

The symptoms of downy-mildew disease of sugar cane are extremely diverse, and are determined largely by the nature of the infection (whether primary or secondary), and the stage in the life cycle of the causal fungus.

The asexual stage is the more common, and concomitant symptoms may be observed at any time of the year. Symptoms peculiar to this stage are more clearly defined following secondary infection than is the case with primary infection, and develop as follows (*see* Fig. 1) : a young leaf unrolling from the spindle first presents a mottled, somewhat mosaic-like appearance at the base; in the course of a few days longitudinal yellowish-white or pale-green streaks develop. Under conditions of vigorous growth these symptoms may be more definite in character and appear as well-defined yellowish streaks, from two to four inches long, and without any apparent mottling. As each successive new leaf unfolds, the affected area gradually extends further from the leaf base until finally the whole leaf is involved. From this stage onwards it frequently happens that the leaf streaks are more conspicuous at the leaf tip than at the base. Leaf streaks are located between the major vascular bundles; they are straight, and, when first formed, fairly regular in outline and initially are separated from each other by strips of normal green tissue of variable width. They are usually distributed across the whole width of the leaf blade, more or less evenly, although it sometimes happens that streaks are confined to one half of the leaf. Streaks have been observed on the midrib, but not on the leaf sheath. Length of streaks varies from a few inches to almost the whole length of the leaf blade, and they may be continuous or broken; width is generally about one to three millimetres, although this measurement is frequently exceeded and streaks up to one centimetre wide are fairly common on certain highly susceptible varieties, such as B.208. Streaks produced during the winter are much narrower and are only a fraction of a millimetre wide. There is a tendency during winter months for diseased plants not to develop leaf streaks when making new growth, and leaves emerging from the spindle may be either apparently healthy, or else possess only a few short inconspicuous watery-green streaks arising from the leaf base. A return to warmer conditions, with more active growth of the plant, is accompanied by the production of normal leaf streaks.

With increasing age, the colour of streaks changes first from greenish-yellow to a more definite yellow, then to mottled reddish-brown, and finally to a uniform dark-red colour. Under conditions extremely favourable for the development of the fungus, the streaks tend

to lose their regularity of outline and, especially towards the leaf tip, fuse to form large irregular yellow or red areas, so that a general discoloration of the whole canetop results. (It is not known to what extent the reddening of streaks is characteristic of downy-mildew disease *per se*; it may be due to the subsequent development of saphrophytic organisms, rather than to *S. sacchari,* since the presence of various species of *Fusarium, Macrosporium,* and *Helminthosporium* is noted on old diseased cane leaves.)

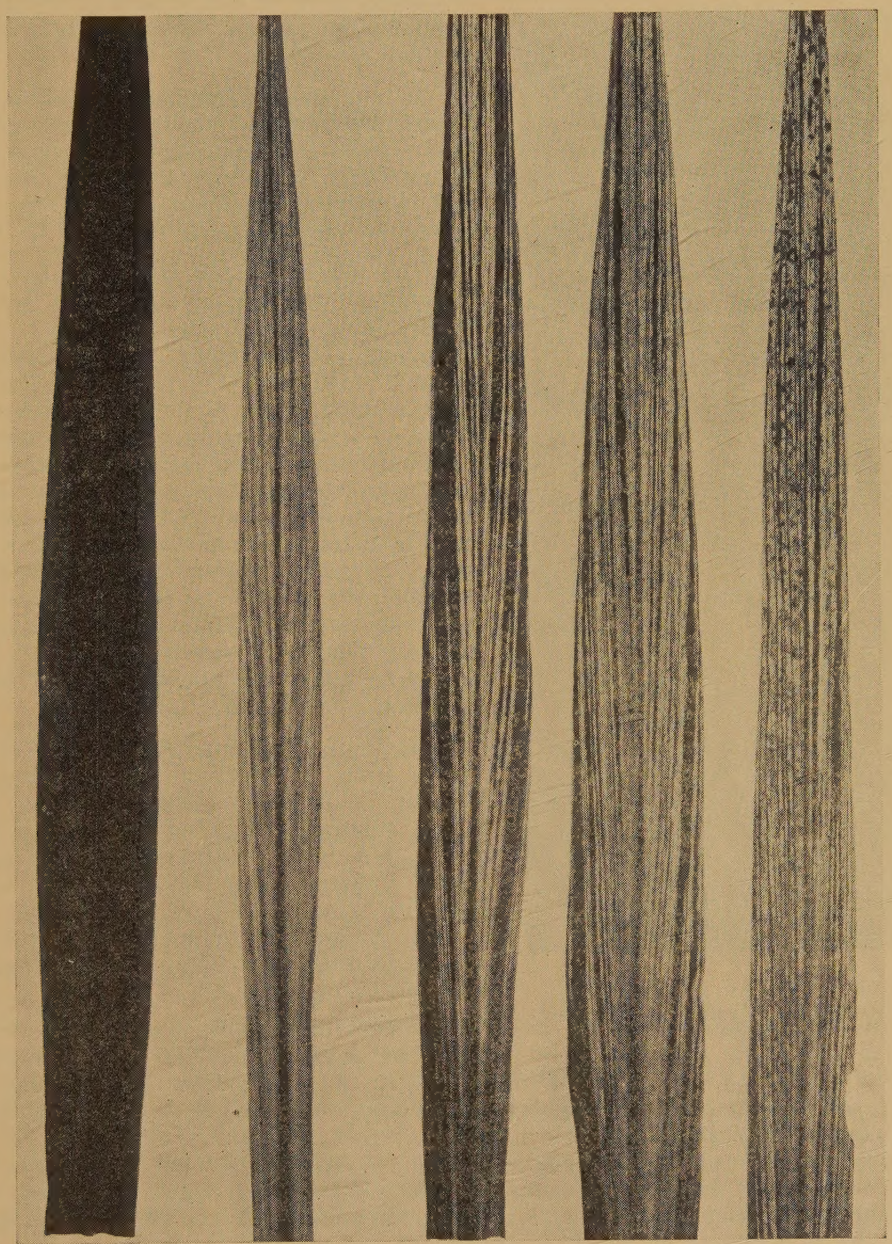

FIG. 1.—Leaves of variety P.O.J.2878 showing typical leaf streaks. The leaf on the extreme left is healthy.

The first leaf streaks are generally developed on young leaves as they unroll from the spindle; they have never been known to develop initially on mature leaves which have already unrolled, and are not present on immature leaves within the spindle. At times the leaf streaks make their first appearance on side shoots developing from the upper nodal buds, these having become infected while the growing point has remained healthy. Disease resistance trials in Queensland show that symptoms of secondary infection may become apparent about two months after planting, or five or six weeks after germination; the period which elapses before the appearance of leaf streaks is, however, largely dependent on varietal susceptibility and prevailing weather conditions.

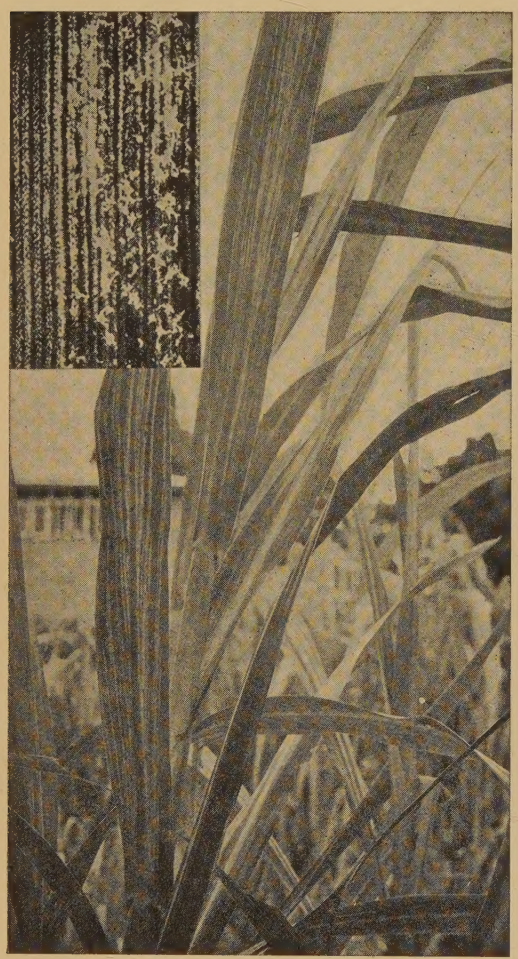

FIG. 2.—Leaf streaks on sugar cane. Inset:—Appearance of "mildew" on under surface of leaf (magnified).

In the case of primary infection, symptoms are usually obvious immediately after germination. Leaf streaks are less clearly defined than is customary with secondary infection and, especially with highly susceptible varieties, the fusion of streaks is more common.

The general appearance of plants in which the asexual stage of the fungus has become established varies according as infection is primary or secondary. In the former instance, there is a pronounced stunting of the plant, and stalks, if any, are extremely thin; in the latter, when only odd shoots of a stool may be obviously infected, these shoots are often a few inches taller than the adjacent canes, so that they stand out prominently in the field, and are conspicuous on account of the general discoloration of the entire top. Leaves of such elongated shoots are somewhat sparse, although they do not split, and canes are again more slender than normal. With the approach of maturity there is frequently swelling of buds along the whole stalk, accompanied by the development of side shoots from the upper part.

Microscopic examination of leaf tissue reveals the presence of mycelium in the mesophyll tissue between the major vascular bundles. This point will be discussed in greater detail in a subsequent paper, but it is evident that discoloration is a result of the invasion by the mycelium, since no difficulty is experienced in finding large quantities in such tissue. Mycelium is also present in the normal green tissue between streaks, or immediately adjacent to them.

Under favourable conditions the development of leaf streaks is accompanied by the nocturnal production of a fine white "mildew," which, on microscopic examination, is found to consist of the conidia and conidiophores of *S. sacchari* issuing from the stomata. The "mildew," when freshly produced, has a soft, velvety appearance, "off-white" in colour; it changes to a fine powder on drying (Fig. 2). Spore production occurs usually on the lower leaf surface, but under hot, wet, extremely humid conditions, it may be observed on the upper surface as well, although this is rare. When climatic conditions are suitable, sporulation accompanies the appearance of the first symptoms, the conidia developing on the discolored areas; as the amount of discolored tissue increases, so also does heavier conidial production take place. On the very young leaves of shoots, in which the disease is well established, sporulation is first apparent towards the leaf tip, but in a very short time it occurs over

almost the whole of the upper leaf blade. Under favourable conditions conidia are produced before the young leaf is fully unrolled from the spindle; they may also be produced in considerable quantities before the leaf streaks become clearly defined. Sporulation is not confined entirely to the discolored areas, although it does not occur on leaves or on sections of leaves from which streaks are entirely absent; that is to say, in the case of a mature leaf with streaks extending from the leaf tip to about four inches from the ligule (which frequently occurs) conidia are produced over the whole of the discolored area, but not at all at the base. Likewise, there is considerably greater conidial production on areas of leaves where streaks are numerous, than on those areas where streaks are comparatively sparse. Sporulation has not been observed on any part of the plant other than the leaf blade.

Sporulation is a nightly occurrence under favourable conditions, and continues on a leaf while it remains attached to the plant. The quantity of conidia produced on a particular leaf each night increases in proportion to the amount of discolored leaf tissue; after the maximum area of discoloration is attained, there follows a gradual reduction in the nightly production of spores. Temperature conditions favourable for sporulation are obtained in Queensland over the period from September to May.

Fig. 3.—Abnormal elongation of stalks (''jump-up'') and sparse leaf development characteristic of the oospore stage of the fungus. Variety, B.208.

The sexual or oospore stage of *S. sacchari* develops in late autumn and winter and is often associated with abnormal growth made by certain stalks in diseased stools. The internodes of such stalks increase rapidly in number, are thinner than usual, and in some cases are longer, so that stalks affected in this manner stand out high above the tops of neighbouring canes (*see* Fig. 3); some such stalks may be twice as long as surrounding healthy stalks; this stage was known as ''jump-up'' by Kanaka labourers in the field and the term is still used. The stalks are light, brittle, and watery, and are changed so much in appearance that varieties are often unrecognizable. The leaves, fewer in number and much shorter than usual, frequently fail to unfold normally, and cling together at the tips, wither, and become twisted and shredded (*see* Fig. 4). Examination of the shredded leaves reveals large numbers of large, brown oospores developed within the mesophyll tissue, which breaks down as the development of the oospores proceeds. Shredding commences at the tips and frequently extends for considerable distances along the leaf blade. The oospores are frequently large enough to be just visible to the naked eye, and are easily detected with a hand lens. Bell [**4**, 1929] estimated that oospores were produced ultimately on about 10 per cent. of the diseased stalks of the variety B.208 in the Lower Burdekin area. Canes affected with this stage of the disease are very shortlived, and usually die before the end of winter or in early spring. Miyake [**28**, 1911] also has reported the presence of this abnormal elongated growth in Formosa, but has stated that the varieties Mauritius Guingham and Rose Bamboo grow abnormally thick.

Leaf twisting and shredding, with the production of oospores, may also be observed on short stunted shoots resulting from the planting of infected setts. In this case, oospores are usually borne on all shoots in the stool without the development of any ''jump-up'' stalks.

Transmission.

The production of conidia of *S. sacchari* is possible in all parts of the Queensland cane belt between September and May, as already stated, but the most favourable period is during December, January and February. The conidia are discharged from the conidiophores when mature and either fall, or else are carried by light winds or raindrops to adjacent plants. In the presence of moisture the conidia germinate immediately, putting forth a germ tube into which the whole contents of the spore are discharged. They are extremely sensitive to drying and cannot withstand sunlight or, in the absence of high humidity, strong winds. Even under favourable conditions they remain viable for only a few hours, so that infection, when it occurs, must be immediate. Field observations indicate that the distance a spore may travel, while still retaining its viability, is not usually more than a quarter of a mile, but observed cases of transmission over greater distances are not rare.

FIG. 4.—Leaf shredding and twisting associated with the development of oospores within the leaf tissues.

The nodal buds or "eyes" are the only part of the sugar-cane plant yet proved to be sensitive to infection, and bud infection is easily demonstrated if setts are stripped free of trash and exposed for a night under diseased plants on which spores are being produced. The subsequent planting of such setts usually results in a high percentage of diseased stools. Moreover, the frequency with which diseased stools arise from the planting of setts taken from apparently healthy plants, and the frequent development of diseased side-shoots on stalks with apparently healthy tops, also provides evidence that bud infection under natural conditions is the general rule. It has also been noted (*see* page 112) that the early symptoms of secondary infection appear at the base of unfolding leaves, thus indicating that invasion of the leaf tissue by the fungous mycelium has proceeded upwards through the region of the growing point.

There is no evidence that the parasite may gain entry and establish itself in developed leaves. Miyake [**28,** 1911] endeavoured, without success, to induce leaf infection by setting diseased leaves in a moist cylinder in such a manner that spores were deposited naturally on the leaves of young plants. The writer has also conducted experiments on similar lines, with negative results, while North [**30,** 1910-5] succeeded only in producing very small brown lesions which did not extend further. If leaf infection took place naturally, a considerably greater rate of secondary infection than ever observed would be expected, in view of the enormous number of conidia produced during a night by a diseased plant and the area of adjacent leaf surface on to which these conidia must fall. Localised areas of infection on individual leaves would likewise be expected to follow direct leaf infection, but these have never been observed. The first symptoms are always observed on immature leaves unrolling from the spindle; they are never seen on mature unrolled leaves.

Whether or not the growing point or very immature leaves within the spindle are susceptible to infection has not been established, although secondary infection of very small potted seedlings has been observed, and secondary infection of young vegetatively propagated cane, on which no nodal buds have appeared above ground, is fairly common. In regard to the question of direct growing-point infection, however, the probability of conidia being washed down the spindle to the growing point of anything other than a very young seedling, is considered unlikely in view of the compression of young leaves in this region. Confirmatory experimental work in regard to immature leaf and growing-point inoculation has not been attempted but the field evidence suggests that infection may be possible through one or other of these places of entry.

The part played by oospores of *S. sacchari* in perpetuating the disease in the field is also as yet undetermined. As will be described in a later paper, numerous attempts (with varied technique) to germinate oospores have failed; attempts to induce infection of the variety P.O.J.213 by planting in sand flats containing oospore infected trash, or by inserting oospores under the bud scales before planting, were also unsuccessful. This failure was somewhat surprising in view of the ease with which oospore germination and infection of the host is achieved with the closely related *S. graminicola* [**25,** 1928] and *S. sorghi* [**46,** 1932].

Dissemination of the disease by mechanical means is considered highly improbable. In view of the nocturnal production of conidia, their extremely delicate structure, and their short period of viability, the transportation of infective spores from one part of a field to another by such agencies as men, animals, or implements, is unlikely. In an experiment involving mechanical transmission, plugs of cane were removed with a cork borer from a stalk of diseased P.O.J.2878 containing abundant mycelium of *S. sacchari*, and then introduced into 25 setts of each of two susceptible varieties, Co.419 and P.O.J.213. The points of inoculation were waxed and the setts planted. All setts germinated and the stools were kept under observation for a year; all remained healthy.

The spread of the disease is greatest in summer, and young plants are more easily infected than more mature ones. The time of ratooning markedly affects the extent of infection, since cane ratooned late in the harvesting season is much more readily infected than cane ratooned early. This is especially striking with varieties that are generally considered fairly resistant to the disease, such as P.O.J.2725; such varieties when ratooned early develop little, if any, infection, but if late ratooning is practised a high percentage of diseased stools may result. Confirmation of field observations was obtained in an experiment where 60 setts of P.O.J.2725 were inter-planted with diseased cane in the Pathology Plot in late September, 1939. After five months growth no sign of the disease was apparent; 15 stools were then ratooned, while all stalks of a further 15 were cut back to a few inches above the growing point, 30 stools being left as controls. Three weeks later three ratooned stools were diseased, and within two months this total had increased to eight; one control stool developed symptoms but these did not become apparent until some months later; no disease symptoms appeared in the cane cut back to a point above the growing point.

Abundant mycelium of *S. sacchari* may usually be found within the tissue of diseased stalks, and setts taken from diseased plants usually produce diseased stools. Instances have been noted however, when both healthy and diseased plants have resulted from obviously diseased stalks, due to the sometimes uneven distribution of the mycelium within the stalk, and its consequent failure to invade certain buds, or to the fact that the mycelium had not yet extended downwards to the butt of the stalk.

2. Alternate Hosts of *Sclerospora sacchari.*

Introduction.

The question of the existence of alternate hosts of organisms parasitic on sugar cane has always been considered of importance by the Queensland Bureau of Sugar Experiment Stations. Cottrell-Dormer [**8,** 1932], in his studies on red-stripe disease, demonstrated that *Phytomonas rubrilineans* Lee *et al.* may also infect maize, sorghum, broom millet (*Sorghum vulgare* Pers.), Sudan grass (*S. sudanense* Stapf), Johnson grass (*S. halepense* L. Pers.), *S. verticilliflorum* Stapf, and *S. plumosum* Beauv. Hughes [**15,** 1939] investigated the host

range of *Bacterium vasculorum* (Cobb) Greig-Smith (the causal organism of gumming disease), and found that maize, sorghum, *Sorghum verticilliflorum,* Sudan grass, Johnson grass, Para grass (*Brachiaria mutica* (Forsk.) Stapf), and elephant grass (*Pennisetum purpureum* Schum.) may also harbour this parasite. The writer has found that *Ceratostomella paradoxa* Dade (the causal organism of pineapple disease of sugar cane), is likewise parasitic on pineapples and bananas, and that a species of *Pythium* parasitic on sugar cane in Queensland is equally parasitic on Sudan grass.

The main practical consideration in such studies is the extent to which the alternate hosts of various cane-disease producing organisms may be responsible for their maintenance. Undoubtedly, the cultivation of resistant varieties affords the most satisfactory means of disease control, and it is the policy of this Bureau to test all new varieties in disease resistance trials before their release for planting can be considered. It is largely by this means that the very great number of serious sugar-cane diseases which occur in Queensland are kept under control. The emphasis on the use of resistant varieties is a natural concomitant of the small-farm system prevailing in Queensland, the annual crop of 5,500,000-6,000,000 tons of sugar cane being produced by some 8,000 growers, each harvesting an average area of about 30 acres per year. It will be recognised that the problems of disease control in such circumstances are much greater than would be the case if all cane were grown on large plantations, each with a staff capable of dealing with the particular disease problem.

In the event of a disease outbreak suddenly assuming proportions so serious that the existence of any standard cane variety becomes threatened in consequence thereof, it may be considered desirable to establish a lower quality but disease resistant variety until such time as the disease is brought under control. As soon as this is accomplished, a return to the old commercially profitable variety becomes a question for consideration, and it is at this point that the existence of alternate hosts may assume great importance. The fact that no disease is apparent in plantings of the main host does not necessarily mean that it has been entirely eliminated; it may be present in some other plant, either wild or cultivated, on which it may perhaps have little effect but which serves to "keep it in reserve", as it were, waiting to attack a susceptible cane variety as soon as such is reintroduced into the district. It follows, therefore, that before proposals for reintroduction are implemented, careful consideration should be given to the possibility of other plants being capable of harbouring the parasite.

Downy mildew is now perhaps the most important sugar-cane disease present in Queensland. The once-serious gumming disease has been brought under control in Southern Queensland as a result of the extensive cultivation of the highly resistant varieties P.O.J.2878 and Co.290, both of which (and especially P.O.J.2878) unfortunately exhibit a high degree of suceptibility to downy mildew. During recent years both these varieties have become increasingly popular with growers in the Central and Southern districts, and it is certain that the extensive cultivation of the former has been largely responsible for the present situation. It became necessary in 1938 to prohibit the planting of P.O.J.2878 in certain tropical areas where the disease was found to be assuming serious proportions and where this is a minor variety; it had also been found that the presence of the disease constituted a barrier to the release of local seedlings which would be otherwise highly suitable for commercial cultivation.

Sclerospora spp. parasitic on other Gramineae.

In addition to *S. sacchari,* some thirteen species of *Sclerospora* have been reported as parasitic on various Gramineae, and an attempt has been made to summarise distribution and host range of these species (Table I). In the compilation of this table, considerable reference was made to the "*Review of Applied Mycology,*" in which it was possible to obtain many references not otherwise available. No claim can be made for completeness, since some references will not have been available to us, particularly among those published prior to the inception of the Review.

Table. I.

HOST RANGE AND DISTRIBUTION OF *Sclerospora* SPECIES.

Species.	Host.	Distribution.	Reference.
S. graminicola ..	*Setaria viridis*	Germany	36
		France	36
		Italy	36
		North America ..	25
		Japan	14
		Russia	3
	S. italica	As for *S. viridis*	
		China	35
		India	45
	Maize (*Zea mays*)	United States ..	25
		Japan	14
		Bulgaria	37
	Pennisetum typhoideum	India	26
		Gambia	19
	S. magna	India	45
		United States ..	55
	Echinochloa crus-galli	Russia	3
	Sorghum sudanense	South Africa ..	1
	Teosinte (*Euchlaena mexicana*)	India	45
		Japan	14
		United States ..	25
	Saccharum sp.	United States ..	25
	Sorghum sp.	United States ..	25
S. sacchari ..	Maize and Teosinte	Formosa	28
		India	22
	Sugar cane	See pages 111, 112	
S. philippinensis	Maize and Teosinte	Philippine Is. ..	49
		India	5
	Miscanthus japonicus	Philippine Is. ..	49
	Sorghum sp., and *Saccharum spontaneum*	Philippine Is. ..	41
S. spontanea ..	Maize, Teosinte, *M. japonicus*, *Saccharum spontaneum*, and *Sorghum* spp.	Philippine Is. ..	50
	Sugar cane and *Saccharum spontaneum* ..	Siam	41
S. sorghi (*S. graminicola*, var. *andropogonis sorghum*)	Sorghum and Maize	India	16, 22, 44
		Egypt	24
	Teosinte	India	46
	Sorghum arundinaceum	Belgian Congo ..	2
S. javanica ..	Maize and Maize-Teosinte hybrids ..	Java	32
S. miscanthi ..	*M. japonicus*, *M. sinense* and sugar cane	Formosa	28
	Sugar cane and *Saccharum spontaneum* ..	Philippine Is. ..	41
	M. japonicus	Philippine Is. ..	52
S. macrospora ..	Sorghum, *Phragmites communis*, Wheat, and Maize	Italy	34, 9
	Wheat	Western Australia	6
	Wheat, *Bromus commutatis*	United States ..	51, 11
	Rice, Panicum, Rye, Oats, Wheat, and Maize	Australia (N.S.W.)	29
	Rice	Bulgaria	10
S. farlowii ..	*Chloris* sp.	North America ..	13
S. magnusiana ..	*Equisetum* sp.	Russia	40
S. butleri	*Eragrostis aspera*	Nyasaland ..	54
S. northi	*Erianthus maximum*	Fiji	52
S. noblei	*Sorghum plumosum* and *Andropogon australis*	Australia (N.S.W.)	53
S. oryzae	Rice	Formosa	38
Unidentified ..	Sorghum	Tanganyika ..	48
	Panicum trypheron	India	23
	Saccharum spontaneum	Siam	41
	Maize and Sorghum	Uganda	39
	Maize, Sorghum, and Sudan grass ..	South Africa ..	42

3. Investigation of Alternate Hosts of *Sclerospora sacchari* in Queensland.

Reasons for Investigation.

Numerous inspections of natural and cultivated grasses in the cane-growing areas of Queensland have been made in the past, not so much in search of downy mildew, but rather in connection with outbreaks of other diseases. Until recent years, however, downy mildew, although widely distributed, has been prevalent only in the Lower Burdekin area, where natural grasses and other crops are very restricted. No case of downy-mildew disease in imported pasture or natural grass was observed until December, 1939, when a variety of Rhodes Grass known locally in Mackay as "Purple-topped Rhodes Grass," [*Chloris barbata* (Sw.) (*inflata*) (L.)] was found to be infected with a species of *Sclerospora.* However, the conidia present were much smaller than those of *S. sacchari,* and the fungus is believed to be a distinct species; morphologically it bears a resemblance to *S. sorghi,* but has not been identified as such.

In December, 1936, leaf streaks suggestive of downy-mildew disease of sugar cane were observed in a volunteer patch of maize (*Zea mays* L.) growing at the Northern Sugar Experiment Station at Meringa; the disease was identified as downy mildew some three or four weeks later, when 75 per cent. of the maize plants were found to be diseased. An inspection of farms in the adjoining area revealed that the disease was present on a number of them, and that in every case the diseased maize was growing in proximity to downy mildew-diseased sugar cane. Similarly affected maize was also observed in the Mackay district in 1937, and near Bundaberg in February, 1938; in these areas also, the diseased plants were growing in close contact with diseased sugar cane. Downy mildew of maize has been reported from these three localities in each succeeding year, but, so far as is known, has not been observed elsewhere in Queensland. It therefore seems probable that the infection was contracted from the diseased cane in these areas. The disease was also reported on sacchaline growing under natural conditions in the Mackay district, in January, 1940.

The greatly extended plantings of the highly susceptible P.O.J.2878 and P.O.J.213, together with the consequential partial adoption of a system of two-year cropping, had very obviously increased the potential and actual importance of downy-mildew disease. This fact, coupled with the discovery of a very similar disease in maize, made it imperative that an investigation of the host range of *S. sacchari* be undertaken without delay.

General Methods and Procedure.

The investigation of transmission of downy-mildew disease from sugar cane to other possible hosts was conducted during the spring-autumn periods of 1938-39, 1939-40, and 1940-41. Except where otherwise stated the work was carried out in the Pathology Plot in Brisbane.

The following species were tested as possible alternate hosts of *S. sacchari*:—

(*a*) *Zea mays* L. (Improved Yellow Dent, Funk's 90 Day, Reid's Yellow Dent, Star Leaming, Golden Beauty, and mixed seed of unknown varieties).
(*b*) *Sorghum* spp. Snow. (Natal 6, Wheatland Milo, American Early Red, Schrock, Hegari, Coleman, Manko, and Atlas varieties).
(*c*) *Panicum maximum* Jacq., var. *coloratum* (Purple-topped Guinea grass).
(*d*) *Brachiaria purpurascens* (Raddi) Henrard (Para grass).
(*e*) *Sorghum halepense* (L.) Pers. (Johnson grass).
(*f*) *Panicum maximum* Jacq. (Guinea grass).
(*g*) *Echinochloa frumentacea* Link. (Japanese millet).
(*h*) *Sorghum sudanense* Stapf (Sudan grass).
(*i*) *Pennisetum purpureum* Schum. (Elephant grass).
(*j*) *Pennisetum typhoides* Stapf and Hubbard (Pearl millet).
(*k*) *Euchlaena mexicana* Schrad. (Teosinte).
(*l*) *Chloris gayana* Kunth. (Rhodes grass).
(*m*) *Sorghum vulgare* Pers. (Egyptian wheat).
(*n*) *Erianthus arundinaceum* (Retz.) Jesw.

The experimental plots were laid out in the same manner as sugar-cane downy-mildew disease resistance trials, viz., rows of experimental varieties are planted so that each contacts a row of diseased cane; if the experimental variety is susceptible to the disease, there is no difficulty in securing infection, providing that natural conditions are favourable for trans-

mission. Planting material consisted of roots of *Panicum maximum*, Guinea grass, Para grass, and Elephant grass, setts of *Erianthus arundinaceum*, and seeds in all other cases. When the work was carried out in Brisbane, seeds were sterilized by washing in 95 per cent. alcohol followed by six minutes immersion in a 1:3000 solution of mercuric chloride, under vacuum, after which they were washed in water. In addition, as a check on the freedom from disease of the planting material, controls were planted in a locality isolated from downy-mildew disease. These precautions were not adopted elsewhere as such observations were largely supplementary to work already carried out in Brisbane.

Results.

Maize.

Observations were made on this host in Brisbane over the period 1938-1941.

1. An experiment was commenced on 19th December, 1938, using the varieties Improved Yellow Dent, Funk's 90 Day, and Reid's Yellow Dent; nine hills of each variety were planted, each hill containing three or four plants. On 10th January, 1939, diseased plants were present in eight hills of Improved Yellow Dent, while plants of the other two varieties were also diseased. On 16th January, all hills of Improved Yellow Dent and Funk's 90 Day were infected, in addition to five hills of Reid's Yellow Dent. All hills of the last-named variety were diseased by the end of January, when all plants were removed. At this time, the plants were about eight inches high, and every plant was diseased. The controls remained healthy.

2. A duplicate of the above was planted in the same locality on 2nd February, 1939. Irregular leaf chlorosis was observed on a number of plants of all varieties on 20th February, but the production of conidia was not noticed until a week later, when it was observed on seven hills of Improved Yellow Dent, five of Reid's Yellow Dent, and one of Funk's 90 Day. Every plant of each variety subsequently became diseased, and conidia continued to be produced until early April. These plants were retained until maturity in order that symptoms could be noted throughout the whole growing period. Controls remained continuously healthy.

3. A planting made on 27th December, 1939, contained the same three varieties, with the addition of a sample of mixed seed of unknown varieties; nine hills of each were again planted. On 18th January, 1940, odd plants of Improved Yellow Dent, Reid's Yellow Dent, and the mixed varieties were infected. A week later all hills of all varieties, with the exception of one hill of Funk's 90 Day, were diseased. Subsequently every plant became infected; the controls remained healthy.

4. Seeds of Improved Yellow Dent were planted in four pots on 24th November, 1939, and placed in the vicinity of diseased cane; all plants were infected by 19th December.

5. Six hills of Improved Yellow Dent were planted on 3rd October, 1940, adjacent to diseased sugar cane which had not yet commenced its nightly spore production. Spore production on sugar cane was first noted on 15th October, but the maize remained apparently healthy until 5th December. Thereafter, seven plants in the five hills exhibited symptoms, the plants being about four feet six inches high when the first symptoms appeared.

6. Six hills of Improved Yellow Dent were planted on 7th November, 1940, in the same plot as Planting No. 5. At the time of planting the nocturnal production of conidia on diseased sugar cane was a fairly regular occurrence. By 25th November, the plants were from four to eight inches high and all were diseased.

A study of the weather data for October, November, and December, 1940, revealed that 11·3 inches of rain fell between 15th October and 5th December, 16 wet days being recorded, while conditions of temperature and humidity were generally favourable for spore production and disease transmission. Comparing series 5 and 6, the delay in the appearance of symptoms of the former may possibly be ascribed to either increased disease resistance of the older plants or to a prolonged "incubation" in such plants. Field evidence suggests that maize plants of advanced age are more resistant than seedlings or very young plants (as is also the case with sugar cane), while crop losses in such cases are greatly reduced.

Observations were also made at Bundaberg where the varieties Improved Yellow Dent, Reid's Yellow Dent, Funk's 90 Day, Star Leaming, and Golden Beauty were included in the sugar-cane downy-mildew disease resistance trial for 1939-40, the planting being made on 11th December, 1939. No details of actual numbers of plants were kept, but the maize was planted in a number of small rows totalling approximately three chains of each variety. Infection was noted on plants of all varieties on 5th January, 1940, and by 17th January, practically every plant was diseased.

Fig. 5.—Downy-mildew diseased maize plant, showing stunting, chlorosis, and early malformation. Variety, Improved Yellow Dent.

The foregoing demonstrates that transmission of the disease to maize is readily effected under the same conditions as apply to transmission in sugar cane; that is to say, provided that temperatures remain above 60° F. and that adequate humidity is maintained, the disease spreads rapidly. The means by which the fungus gains entry to the maize plant have not yet been determined, but the readiness with which plants become infected when only a few inches high suggests that infection occurs either through or adjacent to the growing point, or through very young leaves. Young plants are much more easily infected than older ones, and field evidence indicates that fairly mature plants resist infection (*see* also series 5 page 121). In this connection it is noted that Palm [**32**, 1918] was unable to infect maize plants with *S. javanica* when more than three weeks old, while Weston [**49**, 1920] found it possible to infect maize with *S. philippinensis* at any age.

The symptoms of downy-mildew disease of maize caused by *S. sacchari*, while easily recognisable in the field, are somewhat difficult to describe; they usually

FIG. 6.—Characteristic leaf markings developed on several varieties of maize.

bear rather little resemblance to the specific symptoms of the disease in sugar cane. Reference to Table I, giving the world distribution of the various species of *Sclerospora*, indicates that maize may be infected by at least seven of these. Detailed descriptions of symptoms are not available for all species, but the descriptions given by Weston [**49,** 1920; **50,** 1921] for maize infected with *S. philippinensis* and *S. spontanea* are practically identical with those noted in Queensland in maize infected with *S. sacchari*. The very brief descriptions given by Palm [**32,** 1918] and

Butler [**5**, 1913] of the diseases caused by *S. javanica* and *S. maydis* (which has since been proved to be identical with *S. philippinensis* [**47**, 1936]) apply also to the disease in Queensland, except that the dark brown streaks described by Palm and referred to as type C symptom were not observed.

After the infection of very young maize plants with *S. sacchari* the first symptoms appear on the third to the sixth oldest leaves; such plants may be only four to six inches in height. With older plants, symptoms may be observed on any leaf unrolling from the spindle. Early symptoms take the form of chlorotic, yellowish streaks, 2-3 millimetres wide and with fairly even outline; these extend from the base of the leaf blade to the leaf margin and follow the course of the vascular bundles; one or more streaks may be present on a leaf. With increasing age the streaks lose their watery-green appearance and become more yellow, while the development of new leaves is accompanied by a rapidly increasing degree of chlorosis and a streaked mosaic-like pattern is developed, not only on the leaves, but on the leaf sheaths as well. The chlorotic areas are at first somewhat rectangular, but later coalesce to form large pale areas of indefinite shape, and ultimately the young unfolding leaves are almost completely chlorotic. They are then much smaller than the corresponding leaves of healthy maize; they stand out rather stiffly from the stem, are somewhat brittle, and are easily damaged by wind. A typical diseased plant is illustrated in Fig. 5, while leaf markings are shown in Fig. 6.

Fig. 7.—Leaf streaking and malformation of cobs of diseased maize. The male inflorescence has also produced grain.

Under favourable conditions of temperature and humidity, the development of chlorosis is accompanied by the nocturnal production of a fine "mildew" of conidiophores and conidia on the chlorotic areas of the leaf (both surfaces), and on the leaf sheaths. The environmental conditions necessary for conidial production are the same for maize as for sugar cane (*see* page 114).

The chlorotic leaf markings produced on maize often bear a strong resemblance to leaf markings developed in some varieties following intensive inbreeding. Because of this, and because of the extreme variability of downy mildew symptoms, the presence of conidia upon the leaves must be regarded as the only really positive macroscopic indication that the maize is infected with downy-mildew disease.

In plants which do not make an apparent recovery, a very varied array of distortions and malformations subsequently develops. Cobs are small, twisted, and sometimes entirely without grain but more usually with grain imperfectly formed; they are more numerous than on healthy stalks, as many as five cobs per stalk being of fairly frequent occurrence. They are borne much higher up the stem than normally and are frequently associated with the tassel. The cob shank is often considerably elongated, so much so that at times the cob is protruded through the husk and left entirely without covering of any kind. The tassel is often imperfectly formed and may also develop grain, while witches' broom effects of the whole top

have also been observed. Malformations of the stem often result in the upper part being bent over at right angles, about one foot from the top. The degree of malformation depends on the age of the plant when infected, infection of very young plants resulting in severe stunting with almost complete loss of grain, but the infection of half-grown or more mature plants is attended by considerably reduced losses. Types of malformations more commonly observed are illustrated in Figs. 7-9.

Histological examination revealed the presence of mycelium of *S. sacchari* within the seed, a point observed by Weston [**49,** 1920] with maize infected by *S. philippinensis.* The following experiments were therefore conducted in order to determine whether the planting of such seed would result in the development of diseased plants:—

1. Seed from plants of eight unknown varieties, all of which were diseased, was collected in Bundaberg in July, 1939, and twenty hills, each containing four plants of each variety, were planted in December, 1939. The resulting plants were all healthy. A further planting from the same sample was made in February, 1940, and again healthy plants were produced.

2. Seed collected in Brisbane in May, 1940, was planted in Brisbane (in sand flats in the laboratory) on 3rd June, 1940, and in Cairns in a field planting early in July, 1940. The former was kept under observation for a month and showed no sign of infection; the latter was grown to maturity but all plants remained continuously healthy.

3. Seed from diseased plants of the 1940-41 infection trials was planted on 20th February, 1941, but again all the resulting plants were healthy.

It would appear therefore that downy-mildew disease of maize when caused by *S. sacchari* is at least not commonly transmitted through the seed. This observation conforms with that of Peglion [**33,** 1910] who found mycelium in the pericarp of wheat infected by *S. macrospora,* but obtained diseased plants only when the grain was planted before it was fully ripe.

Fig. 8.—Illustrating leaf streaks, malformation, and increased cob production in diseased maize.

Teosinte.

Observations on teosinte were made in Brisbane during the summer of 1939-40. Seeds were planted adjoining diseased sugar cane on 19th January, 1940, the experimental plot comprising nine hills, each containing two or three plants. On 7th February, 19 days after planting, the plants were three or four inches high and were all diseased; their leaves were almost completely yellow and heavy sporulation of *S. sacchari* was occurring on both leaf surfaces. All plants were very stunted and very little growth was made for the next four weeks; the nocturnal production of conidia ceased early in March, and thereafter new leaves produced were of a normal green colour with pale green mottling. In some cases the amount of mottling was reduced with each successive leaf, so that it appeared as if these plants were recovering when leaves almost entirely devoid of mottling were produced. This apparent

recovery was in no case permanent, for in every instance, further mottled and sometimes malformed leaves were produced before the plants attained full growth; there was not, however, any further production of conidia.

Leaves of teosinte infected with *S. sacchari* bear a very strong resemblance symptomatically to those of diseased maize, the general pattern being mottled rather than streaked, with the pale areas often fusing to form large irregular patches. Definite streaks are sometimes produced, however, and these are yellowish and flecked with orange; their width is fairly even (1-2 millimetres) and they are somewhat broken throughout their length. Such streaks bear some resemblance to leaf streaks on sugar cane and sorghum, but are extremely variable in their general appearance. As the plant approaches maturity, new leaves are shorter, and stand out very stiffly from the stem, as illustrated in Fig. 10.

FIG. 9.—Malformation of diseased maize, showing extreme elongation of cob shank and sparse grain production on cob.

The most marked effect of downy-mildew disease on teosinte is one of stunting, the degree of which varies considerably among plants originally infected at the same time. Little stunting was observed in those plants which had exhibited some tendency towards recovery following the cessation of conidial production, but others were markedly dwarfed. No severe malformation was ever recorded, except in the case of the short, topmost leaves. Seed production, although it may have been reduced, was not otherwise adversely affected, even on badly diseased plants.

Sorghum.

Infection trials with sorghum were carried out in Brisbane in 1938-39 and 1939-40, experiments being conducted as under:—

1. Three rows, each containing 30 to 40 plants of each of the varieties Wheatland Milo, Natal 6, and American Early Red, were planted in the Pathology Plot on 19th December, 1938, concurrently with the first maize planting. Twenty-eight days after planting leaf streaks suggestive of downy mildew were observed on two plants of the variety Natal 6, but there was no visible production of conidia. One plant was therefore incubated overnight in a moist chamber, and a sparse production of conidia resulted. Conidia were also produced *in situ* on the other plants on the nights of the 17th and 19th January, but on neither occasion was their production sufficiently heavy to be seen as a mildew with the naked eye. By 25th January, leaf markings were present on two plants of Wheatland Milo, while two more plants of Natal 6 had developed symptoms; scrapings from the lower leaves of these plants were examined microscopically, but no conidia were observed. In early March, when they were about ten weeks old, most of the plants of all three varieties showed leaf symptoms, and on the night of 14th-15th March a few conidia were produced on odd leaves of the varieties Natal 6 and American Early Red, but again there was no visible mildew. (On this occasion the plants were examined at 4 a.m. on the morning of 15th March; rain had fallen throughout the night and there had been heavy conidial production on both sugar cane and maize.) The plants were retained until July, 1939, but no further conidial production was noted.

Before the conclusion of observations on this planting, leaves, leaf sheaths, and inflorescences were examined for the presence of oospores, and dead leaves in which it was thought that oospores might develop were retained in the laboratory for a further six months. No development of oospores was noted at any time.

2. Twenty-four plants of each of the above three varieties were planted on 3rd February, 1939. With the exception of one plant of the variety American Early Red, which developed leaf streaks when five weeks old, all plants grew to maturity without the appearance of leaf symptoms. Spore production was not observed.

3. The grain sorghum varieties American Early Red, Coleman, Schrock, Manko, Atlas and Hegari were planted in late December, 1939. The plants were grown almost to maturity without any development of symptoms and were then ratooned. In the ratoons, indefinite leaf streaks, suggestive of downy-mildew infection, developed on odd plants of American Early Red, Coleman and Atlas. However, sporulation did not occur *in situ* and could not be induced in moist chambers. Attempts to demonstrate the presence of mycelium within the tissue, using Garrett's method [**12**, 1937], also failed.

FIG. 10.—Teosinte: short stiff leaves characteristic of diseased plants.

4. The varieties American Early Red, Schrock, Manko, Coleman, Atlas and Hegari were also included in the sugar-cane downy-mildew disease resistance trials conducted at Bundaberg, Mackay and Meringa in the 1939-40 season. A variety of *S. vulgare,* known locally as Egyptian Wheat, was also planted at Mackay.

The Bundaberg planting was made on 11th December, 1939, when two 15-feet rows of each variety were planted. By 5th January, 1940, there was fairly heavy infection of American Early Red plants, with light but definite production of conidia. Suspicious leaf markings were also recorded on Schrock, but sporulation was not observed. The remaining plants failed to develop symptoms, even after ratooning.

The Mackay planting was made on 18th October, 1939. Only one plant of the variety Coleman developed definite downy mildew, while other plants of the same variety merely exhibited suspicious leaf symptoms.

The Meringa planting was made on 2nd November, 1939. The plants were grown almost to maturity and were ratooned on 20th February, 1940. No symptoms appeared on any plant.

On sorghum, as is the case with maize, the first visible symptom of downy-mildew disease is a single yellowish streak on one of the basal leaves. The appearance of this streak is followed by a gradually increasing amount of chlorosis as further leaves unfold; the leaf pattern becomes streaked rather than mottled, thus more closely resembling symptoms on sugar cane than on maize. Streaks, of which the width varies from one-half to two millimetres, commence near the midrib and extend towards the edge of the leaf blade, following the direction of the vascular bundles. They are yellowish, and are usually mottled with orange-red flecks. Streaks are generally continuous, but may be broken, and on older leaves they alternate with bands of normal green tissue. very young leaves on a plant infected early may be completely chlorotic. Sporulation, when it occurs, is always sparse, and has been observed only on lower leaf surfaces. Plants grow to maturity without malformation and without appreciable stunting. The top of a diseased sorghum plant is illustrated in Fig. 10.

Fig. 11.—Leaf streaks on diseased sorghum. Note similarity to symptoms on sugar cane. (*See* Fig. 1.)

Sudan Grass and Johnson Grass.

Seeds of Sudan grass and Johnson grass were planted on 17th October, 1938, each in three rows of three-feet length. On 16th January, 1939, leaf streaks were visible on two plants of Sudan grass, but there was no evidence of sporulation having occurred. Leaves were incubated overnight in a moist chamber in an effort to induce spore production but the attempt was not successful. On 25th January, one shoot of one Johnson grass plant had also developed suggestive leaf streaks, without spore production, and again the attempt to produce spores under laboratory conditions failed to yield positive results.

Both grasses remained under close observation until 9th February, when they were ratooned. In the period between the first observation of leaf streaks and the date of ratooning there was a considerable increase in the number of shoots of Sudan grass showing leaf streaks, but at no time was spore production observed, nor could the presence of fungus mycelium within stem and leaf tissue be demonstrated. In the ratoons the same streaking was apparent on both grasses, and on the morning of 15th March, after a night when spore production on sugar cane and maize had been particularly heavy and had also occurred on two varieties of sorghum (*see* page 126), a few odd conidia and conidiophores of *S. sacchari* were scraped from leaves of Johnson grass. No further conidia were ever seen, and the plants were retained through the winter until the following spring, when leaves, leaf sheaths, and inflorescences were unsuccessfully examined for oospores.

In November, 1940, there was considerable leaf streaking in a number of volunteer Johnson grass plants in the Pathology Plot in Brisbane. A number of attempts to induce sporulation were again unsuccessful, but careful examination of young stem tissue revealed the presence of traces of a phycomycetous mycelium which may have been *S. sacchari,* but could not be definitely identified as such.

In the above observations it will be noted that conidia were observed on Johnson grass on one occasion only, and, in view of the very small number present, it is possible that these may have been washed on to the leaves from adjacent badly diseased sugar cane, although odd conidiophores appeared to be attached to pieces of epidermis, suggesting that they were developed *in situ.* Because of this unsatisfactory spore production, combined with the paucity of mycelium within the tissue, it cannot be stated definitely that the plants were infected with downy mildew, although the appearance of the leaves suggested very strongly that this was the case. Vigorous growth, lack of sporulation, and the comparatively few leaves showing streaks all suggest that if the mycelium were present at all within the stem, it would be there in only very small quantities, and perhaps difficult to locate. It must also be remembered that spore production on sorghum is by no means a regular occurrence, but takes place only under exceptionally favourable conditions, and the single night on which conidia were found on Johnson grass was one of the few nights when sporulation occurred on sorghum in Brisbane. Much more definite infection of sorghum was observed in districts north of Brisbane, than in Brisbane itself, and such may have proved to be the case with the two grasses concerned, had it been possible to conduct observations in other districts. Although further evidence is necessary before it can be definitely stated that Sudan grass and Johnson grass may function as alternative hosts of *S. sacchari,* such evidenc as is available is strongly suggestive of such behaviour.

The observed leaf streaks were at first yellow, later becoming mottled red and finally entirely red. They were even in outline, about one millimetre wide, and alternated with bands of normal green tissue of the same width. The streaks, which extended along the whole length of the leaf, bore a very strong resemblance in miniature to those on diseased cane plants. Streaks on very young leaves were short (sometimes only a fraction of an inch long) and narrow, giving a mottled effect. Other young leaves were chlorotic, with mosaic-like markings, red mottling, or very small red or yellow streaks. The affected leaves were short in comparison with healthy leaves, and stood out very stiffly from the stem (as is the case with infected maize and teosinte) and shoots on which streaked leaves were present were much reduced in size, the floral parts sometimes failing to develop. Diseased leaves and shoots are illustrated in Figs. 11 and 12.

Other Grasses.

Para Grass, Guinea Grass, Elephant Grass, Japanese Millet, Pearl Millet, and *Panicum coloratum* were planted on the 16th October, 1938, and were grown until late autumn 1939. All were ratooned at least once and, during the 1939-40 summer, odd volunteer plants of the various grasses were observed growing in the vicinity of the experimental plot. All plants remained continuously healthy throughout the period of observation.

Rhodes grass was planted in pots on 21st February, 1940, and grown in the vicinity of diseased cane until June, 1940, but failed to contract the disease.

FIG. 12.—Johnson grass: appearance of streaks on leaves of plants exposed to infection by *S. sacchari*.

Erianthus arundinaceum was included in the sugar-cane downy-mildew disease resistance trial at Bundaberg for the 1939-40 season. Six setts were also planted adjacent to diseased sugar cane in the Pathology Plot in Brisbane in December, 1940. Prior to planting, the setts were exposed for a night under sugar cane on which large numbers of conidia were being produced nightly. No symptoms developed in either planting.

4. Transmission of Downy Mild w from Maize to Sugar Cane.

The foregoing results having demonstrated the ease with which downy-mildew disease may be transmitted from sugar cane to maize, and the profusion with which conidia may be produced on the leaves of diseased maize plants, it remained to be proved that the disease could be transmitted in the reverse direction, from maize to sugar cane.

Downy mildew is fairly readily transmitted to healthy cane of susceptible varieties by the nocturnal exposure of the tender buds of freshly stripped cane beneath the leaves of diseased plants. Upon planting such cane, a proportion of the exposed buds gives rise to diseased plants. Accordingly, 25 setts of healthy cane of the variety P.O.J.213 were stripped free of trash and exposed under diseased maize plants for a night in March, 1939, at a time when large quantities of conidia were being produced nightly. In order to prevent contamination from diseased sugar-cane plants growing in the vicinity, the maize plants were covered overnight by a sheet of thick calico thrown over a frame and with the edges sealed to the ground in such a manner that there could not possibly be any entry of conidia from without. The sugar-cane setts were placed in position during the late afternoon, when no spores left from the previous night's production would be viable. The setts were planted the following morning, and of the resulting stools, five were diseased. Transmission of the disease from maize to sugar cane has also been reported by Miyake [**28,** 1911] who planted healthy cane setts adjacent to diseased maize plants, the resultant cane plants later becoming diseased.

5. Discussion.

The marked susceptibility to downy mildew of most Kassoer derivatives so far tested, has attached a new importance to this disease; it is consequently a fortunate circumstance that its presence is restricted to a few countries in the Western Pacific zone. While the frequently long "incubation" period increases the difficulties of plant selection in the presence of the disease, nevertheless the obvious nature of the symptoms makes its ultimate detection easy; there should be no likelihood of downy mildew in sugar cane penetrating reasonably efficient quarantine defences.

A considerable body of evidence, both direct and indirect, has been adduced in favour of immature nodal bud infection being at least the main method of secondary infection of sugar cane. Such method of infection serves to explain both the lag between infection and development of symptoms in the cane top and the relatively low rate of infection compared with the myriads of spores dispersed. The protection of immature buds provided by the tightly bound leaf sheaths, and the extreme sensitivity of conidia to desiccation, are factors of the utmost importance in making the field control of this disease possible. The inhibition of sporulation and transmission due to low temperatures and/or low humidities experienced during winter and spring serve to make practicable the field control of this disease, by roguing of diseased stools during this period.

Oospore production is profuse in the tropics but we have no tangible evidence that these resting spores have initiated a disease outbreak. Nevertheless, and in spite of failure to induce germination of these spores in the laboratory, they must continue to be regarded as potential sources of infection. For this reason the burning of infected crops before harvesting is retained as a disease control measure.

The experiments here recorded demonstrate that the cultivation of maize and teosinte in the vicinity of downy mildew-diseased sugar cane results, under suitable conditions, in the ready infection of these two crop plants. Infection of commercial varieties of sorghum, though readily demonstrated, was not nearly so intense. On the other hand the fodder and weed grasses most commonly found in and around Queensland cane fields (*see* page 120) appear highly resistant or immune to attack by *S. sacchari,* only two (Johnson and Sudan grasses) having possibly become infected. Since these experimental results are in accord with such field observations as are available, it is now possible to formulate some picture of the role likely to be played by alternate hosts in the persistence and transmission of downy-mildew disease of sugar cane.

FIG. 13.—Johnson grass: healthy shoot (left), and shoots suspected of being diseased. Note shortening and stiffening of upper leaves which is a characteristic of downy-mildew disease of sugar cane on other proved hosts.

Maize is, undoubtedly, the most seriously affected alternate host; all five named varieties, together with the mixed progenies, were readily infected and diseased leaves produced abundant conidia under suitable meteorological conditions. In contrast to sugar cane, the rapidity with which a whole field of maize may become infected is startling; plants from known disease-free seed may become infected to the extent of 100 per cent. within a couple of weeks of the onset of weather suitable for aerial transmission.

This disease has not been observed to attack maize in localities where sugar cane is not grown, nor has it been observed on maize grown in those sugar-cane districts where downy mildew has not been reported—at least in recent times. Maize is, of course, a strict annual and several plantings of seed taken from cobs produced on diseased plants, and bearing grain in which mycelium of the causal fungus could be detected, failed to yield diseased plants. At the same time, on the basis of very considerable field experience, some of the Field Inspectors

employed by Sugar Cane Disease Control Boards feel that there is reasonable evidence that disease outbreaks may be initiated by the planting of diseased seed. Be that as it may, however, it is agreed that such form of transmission is at least very uncommon. The production of oospores or other type of resting spore has never been observed in the tissues of diseased maize plants.

It would therefore appear reasonable to assume that maize is at least of little importance in the perpetuation of downy-mildew disease of sugar cane. Conversely, it is potentially a serious factor in rapid dissemination in the presence of an existent source of infection. The prohibition of the planting of maize in the vicinity of diseased sugar cane in the Bundaberg district has been followed by a marked reduction in the incidence of the disease and, while direct correlation cannot be proven, it is believed that the elimination of this alternate host has been an important contributory factor in this reduced incidence. It is, of course, recognised that the passage of a parasite through an alternate host may result in a greater or less reduction in virulence. Unfortunately, facilities are not available for the evaluation of this possibility under adequately controlled conditions, but field evidence suggests that there is at least some degree of attenuation.

Since young plants are more susceptible than older plants, it follows that earlier planted maize is likely to be of less danger in the presence of the disease.

Much of the foregoing applies equally to teosinte but this crop is not grown commercially in Queensland, and its susceptibility to downy mildew may therefore be regarded as of academic interest only.

Sorghum is likewise an annual, although, unlike maize, it is ratooned to some extent. It is far less susceptible than maize but, on the other hand, the symptoms are far less obvious and the presence of the disease less readily detected. From the latter point of view, and because of the much smaller crop loss, it may equal maize in importance as an alternate host; a farmer who has had experience of acute downy mildew in maize usually requires little persuasion to discontinue planting this crop. Fortunately, conidial production is much less abundant on sorghum.

The failure of both Johnson grass and Sudan grass to produce conidia or oospores on or in supposedly diseased leaves suggests that they are not likely to be of importance in disease dissemination. However, since these two hosts were not studied under tropical conditions, it is evident that further investigation is necessary before final conclusions can be drawn; this is particularly so in the case of Johnson grass, since it is a perennial, propagated vegetatively.

Great variability of symptoms between plants of a single host, and between different host plants, was noted throughout the observations. Generally speaking, there was a strong resemblance in symptoms developed in the closely related maize and teosinte, but as between the other hosts investigated such resemblances were not marked. Leaf mottling and general chlorosis are conspicuous features in maize and teosinte, clearly defined leaf streaks being found, as a rule, only in the earliest stage of the disease; the reverse applies to sugar cane and sorghum. A symptom common to all hosts was the shortening and stiffening of the topmost leaves as the disease progressed, this being a general characteristic of plants infected by species of *Sclerospora*. A further point of interest is the fact that the symptoms as developed in maize, following infection by *S. sacchari*, very closely resemble those described in respect of maize infected by *S. philippinensis* Weston [**49,** 1920], *S. spontanea* Weston [**50,** 1921] and *S. javanica* Palm [**32,** 1918].

The relationship of *S. sacchari* to other species of the same genus has received some consideration on the bases of morphology, host range and symptoms on common hosts. Many of the early descriptions of species are quite inadequate and the writer is in agreement with Weston [**49,** 1920] that further detailed investigation may demonstrate that numbers now classified as distinct species are so closely related as not to have warranted the establishment of new species. Further aspects of these relationships will be discussed in a later paper.

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